MOLECULAR PLANT PATHOLOGY (2004) **5**(3), 157–166 DOI: 10.1111/J.1364-3703.2004.00218.X

Pathogen profile

Sugarbeet leaf spot disease (*Cercospora beticola* Sacc.)†

JOHN WEILAND[1,*] AND GEORG KOCH[2]

[1]*United States Department of Agriculture, Agricultural Research Service, Northern Crop Science Laboratory, Fargo, ND 58105, USA*
[2]*Strube-Dieckmann, A. Dieckmann-Heimburg, Postfach 1165, 31684 Nienstädt, Germany*

SUMMARY

Leaf spot disease caused by *Cercospora beticola* Sacc. is the most destructive foliar pathogen of sugarbeet worldwide. In addition to reducing yield and quality of sugarbeet, the control of leaf spot disease by extensive fungicide application incurs added costs to producers and repeatedly has selected for fungicide-tolerant *C. beticola* strains. The genetics and biochemistry of virulence have been examined less for *C. beticola* as compared with the related fungi *C. nicotianae*, *C. kikuchii* and *C. zeae-maydis*, fungi to which the physiology of *C. beticola* is often compared. *C. beticola* populations generally are not characterized as having race structure, although a case of race-specific resistance in sugarbeet to *C. beticola* has been reported. Resistance currently implemented in the field is quantitatively inherited and exhibits low to medium heritability.

Taxonomy: *Cercospora beticola* Sacc.; Kingdom Fungi, Subdivision Deuteromycetes, Class Hyphomycetes, Order Hyphales, Genus *Cercospora*.

Identification: Circular, brown to red delimited spots with ashen-grey centre, 0.5–6 mm diameter; dark brown to black stromata against grey background; pale brown unbranched sparingly septate conidiophores, hyaline acicular conidia, multiseptate, from 2.5 to 4 μm wide and 50–200 μm long.

Host range: Propagative on *Beta vulgaris* and most species of *Beta*. Reported on members of the Chenopodiaceae and on Amaranthus.

Disease symptoms: Infected leaves and petioles of *B. vulgaris* exhibit numerous circular leaf spots that coalesce in severe cases causing complete leaf collapse. Dark specks within a grey spot centre are characteristic for the disease. Older leaves exhibit a greater number of lesions with larger spot diameter. During the latter stage of severe epiphytotics, new leaf growth can be seen emerging from the plant surrounded by prostrate, collapsed leaves.

Control: Fungicides in the benzimidazole and triazole class as well as organotin derivatives and strobilurins have successfully been used to control Cercospora leaf spot. Elevated levels of tolerance in populations of *C. beticola* to some of the chemicals registered for control has been documented. Partial genetic resistance also is used to reduce leaf spot disease.

* *Correspondence*: E-mail: weilandj@fargo.ars.usda.gov

†Mention of a trademark or proprietary product does not constitute a guarantee or warranty of the product by the USDA or imply approval to the exclusion of other products that may also be suitable.

INTRODUCTION

Sucrose from sugarbeet is an important dietary supplement worldwide. With production at ~35 000 000 tonnes in 2002, just less than one-third of world sucrose supplies are derived from sugarbeet (Anon., 2003). Of this, nearly a half originated from countries in the European Union. Beet sugar production is distributed across continental regions characterized by temperate climates, complementing cane sugar production in more tropical climes.

Cercospora leaf spot caused by *Cercospora beticola* is considered to be the most destructive foliar pathogen of sugarbeet in the world. The first report characterizing Cercospora leaf spot disease was published by Saccardo (1876). Before the beginning of the 20th century, investigators gave various names to the fungus causing leaf spot diseases of sugarbeet, most of which by description probably were *C. beticola* (Chupp, 1953). Warm, humid growing regions are most acutely affected by Cercospora leaf spot and constitute greater than 30% of the area under sugarbeet cultivation. Producers in such areas must diligently apply fungicides to varieties possessing moderate to high genetic resistance to the disease in order to bring the crop to maturity (Meriggi *et al*., 2000; Windels *et al*., 1998; Wolf and Verreet, 2002). Without such measures, the leaf canopy of sugarbeet fields can be destroyed by outbreaks of *C. beticola*, resulting in complete loss of the crop (Fig. 1; Duffus and Ruppel, 1993; Rossi *et al*., 2000).

Resistance to Cercospora leaf spot in sugarbeet has been described as quantitatively inherited and rate limiting with respect to disease development (Rossi *et al*., 1999; Smith and Gaskill, 1970). Although this resistance to *C. beticola* has proven to be effective in both North America and Europe, it nonetheless exhibits low heritability (Smith and Ruppel, 1974); varieties bred for

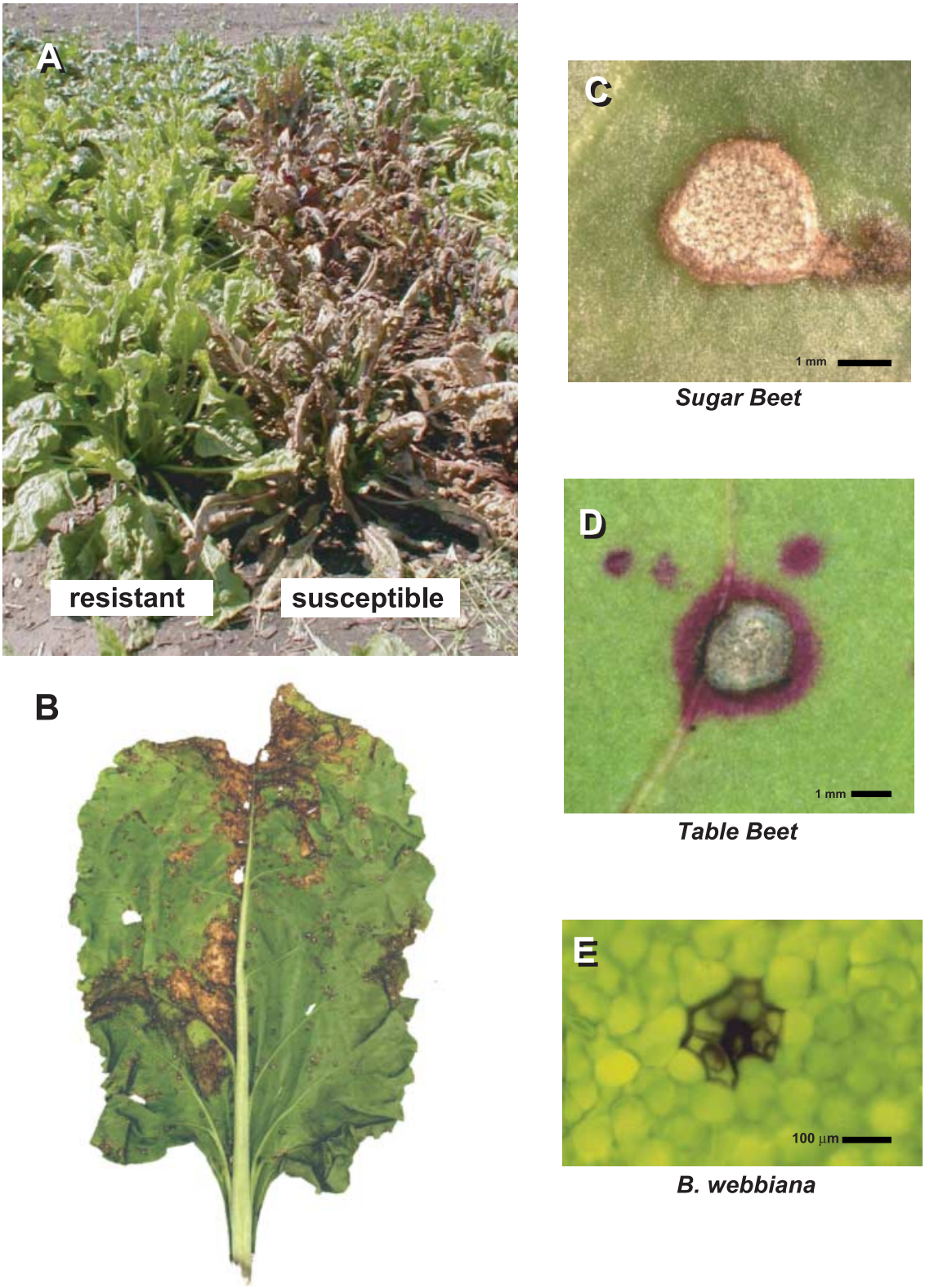

Fig. 1 Symptoms of *C. beticola* on leaves of various *Beta* species. Panels A–C illustrate reactions on sugarbeet and panel D shows the reaction on table beet. The lesion size in A–D contrasts with that on *B. webbiana*, in which only a few cells exhibit reaction to attempted penetration by the fungus.

Cercospora resistance can still exhibit leaf spot if climactic conditions favourable for the disease occur. For this reason, the timely application of fungicides in conjunction with forecasting models that predict the likelihood of Cercospora infection has become an important complement to genetic resistance in leaf spot control (Windels *et al*., 1998; Wolf and Verreet, 2002). Compounding this issue is the well-documented occurrence of fungicide tolerance in *C. beticola* populations (Ioannidis and Karaoglanidis, 2000). As a consequence, the control of leaf spot disease necessitates the judicious rotation of fungicide chemistries as a means of preventing or forestalling the development of resistant strains or reducing their prevalence in the populations.

CAUSAL ORGANISMS

Taxonomy

The organism *Cercospora beticola* is an imperfect filamentous fungus with no known sexual stage (Fig. 2; Chupp, 1953; Duffus and Ruppel, 1993). *C. beticola* infects species of the genus *Beta*, an important taxonomic characteristic, and a number of species in the Chenopodiaceae, including members of the genera *Spinacea*, *Atriplex* and *Amaranthus*. It has been noted that leaf spotting fungi on weed species having needle-shaped, hyaline spores have occasionally, if probably hastily, been classified as *C. beticola*. This often could prove erroneous as leaf spot diseases caused by other species of *Cercospora* have been characterized on weeds in the presence of, and on crop species in rotation with, leaf and root beet crops (Chupp, 1953). Although limited host-range studies with *C. beticola* have been performed, systematic investigation confirming the host range of Cercosporoid fungi isolated from the leaf spots of beet, an important characteristic in *Cercospora* taxonomy, has not to our knowledge been reported. Production by *C. beticola* and related species of the phytotoxin cercosporin has been a useful taxonomic tool in a broad sense, but is too variable in its expression in culture between strains of a species, as well as across culture media formulations, to be relied on for fine taxonomic separation (Goodwin *et al*., 2001).

No sexual stage has been found for *C. beticola*, unlike several *Cercospora* fungi for which a *Mycosphaerella* teliomorph has been characterized. None of the *Cercospora* species considered to be monophylectic with *C. beticola* based on rDNA sequences possesses a known teliomorph, suggesting that this function may have been lost during evolution of the group (Goodwin *et al*., 2001). Nevertheless, hyphal anastomosis or an elusive mating system may promote genome exchange in *C. beticola*, contributing to genetic diversity within natural populations. Surveys revealing diversity in fungicide resistance in populations of *C. beticola* under fungicide pressure are legion (Ioannidis and Karaoglanidis, 2000). Comparison by amplified fragment length polymorphism (AFLP) analysis of single-spore isolates of *C. beticola* in two independent studies in Europe and the USA indicate substantial genetic variation in natural populations (Große-Herrenthey, 2001; Weiland *et al*., 2001). Moreover, subculture of *C. beticola* in the laboratory has been reported to result

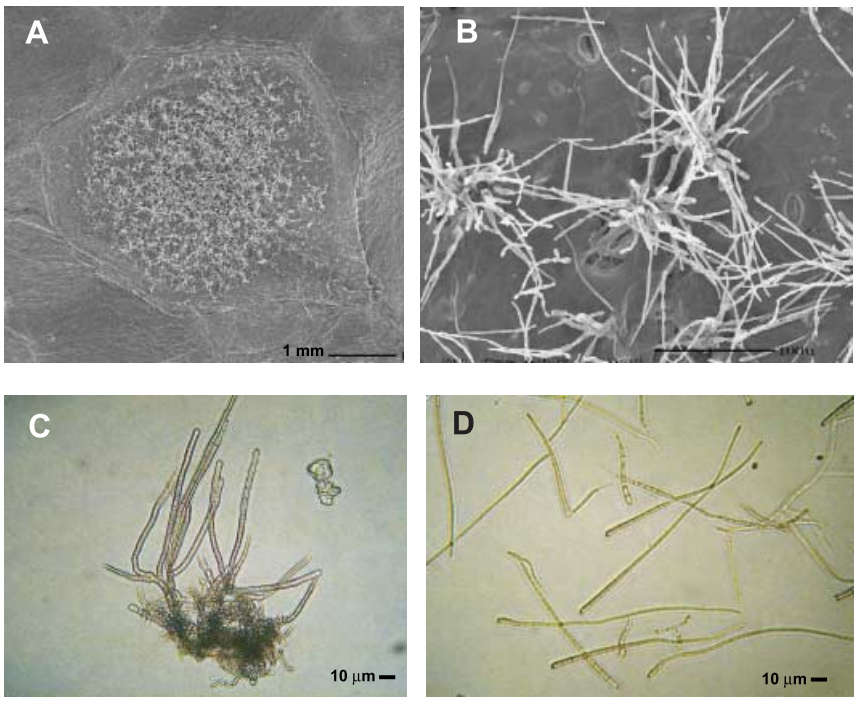

Fig. 2 Micrographs of *C. beticola* conidiophores and conidia. Scanning electron micrographs in panels A and B illustrate the topology of these structures within disease lesions. Accompanying these are light micrographs of conidiophores (C) and conidia (D).

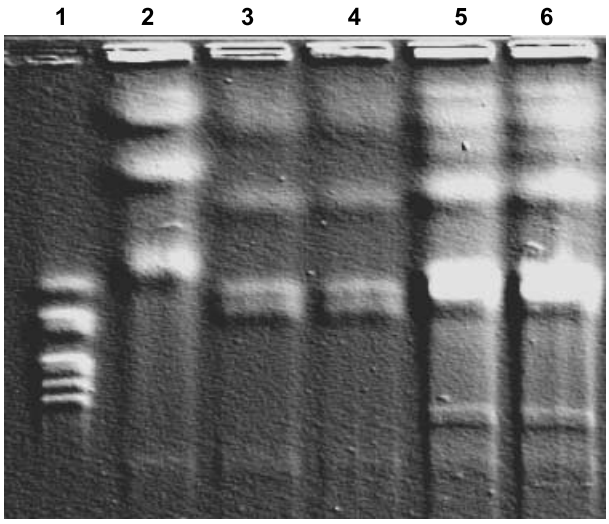

Fig. 3 Chromosome separation by pulse-field gradient electrophoresis of *C. beticola* 98-23A (lanes 3–4) and an isolate from repeated subculture of 98-23A (lanes 5–6). Chromosomes of *H. wingei* (lane 1) and *S. pombe* (lane 2) are included as controls. Note changes in the size and number of chromosomes between the two *C. beticola* isolates.

in chromosome rearrangements as assayed by pulse-field gradient electrophoresis (Fig. 3). This apparent genome plasticity in *C. beticola* combined with limited sequence diversity in rDNA regions of related *Cercospora* species contributes to the present confusion in the taxonomy within this genus (Goodwin *et al.*, 2001). Although both growth morphology in culture and virulence have been used as characters for the grouping of *C. beticola* isolates (Ruppel, 1972), use of this information in the description of unique field strains has not gained acceptance. One exception is the description by Whitney and Lewellen (1976) of the field isolates C1 and C2. Strain C2 is distinguished from C1 by its ability to induce a low-virulence 'fleck' reaction on inoculated sugarbeet possessing the *Cb* resistance gene. Strain C2 also exhibited higher virulence than strain C1 on sugarbeet varieties lacking the *Cb* gene. Although race C2 may exist within larger populations of *C. beticola*, races of the C1-type that are not affected by presence of the *Cb* resistance gene appear presently to dominate in regions growing sugarbeet (Duffus and Ruppel, 1993; Lewellen and Whitney, 1976).

Symptoms and phytotoxins

Characteristic symptoms of the infection of *Beta* species by *C. beticola* include the random distribution of leaf spots of typically 0.2–0.5 cm in diameter across the surface of mature leaves (Fig. 1; Duffus and Ruppel, 1993). Unlike many other leaf spot pathogens that necrotize from pin-point lesions and expand outward, lesions produced by *C. beticola* involve the near-simultaneous collapse of cells in an area many millimetres in diameter. This is due to the fact the *C. beticola* enters the host through stomata followed by initial colonization of the tissue in an asymptomatic manner (Feindt *et al.*, 1981a; Steinkamp *et al.*, 1979). After tissue collapse has occurred, the lesion often becomes encircled by a characteristic reddish-brown ring. Lesions can be observed on leaves and leaf petioles. Although lesions can expand after initial tissue collapse, the increase in necrotic area on the leaf surface is due primarily to an increase in the number of lesions on that surface. Ultimately, a combination of high lesion number and the accumulation in the leaves of phytotoxins induces complete leaf senescence.

Resistant varieties of *Beta vulgaris*, including sugarbeet, table beet and chard, exhibit reduced lesion size, reduced lesion numbers on infected leaves and reduced conidial production per unit area of lesion (see below). As mentioned above, small 'fleck' lesions are induced by *C. beticola* race C2 on germplasm harbouring the *Cb* resistance gene, reminiscent of a hypersensitive response. Interestingly, inoculation of *Beta webbiana*, a wild beet in the section *Procumbentes*, with *C. beticola* results in the rapid (2–3 days post-infection, d.p.i.) development of reddish coloration in the cells immediately surrounding the penetrated stomata (Fig. 1; Carels *et al.*, 1990). No production of characteristic grey necrosis is observed at these sites, suggesting that the reaction develops in a manner analogous to, but distinct from, a hypersensitive response.

Most *Cercospora* species, including *C. beticola*, are considered to be necrotrophs, producing low-molecular-weight phytotoxins and hydrolytic enzymes that debilitate cells in advance of fungal growth. The mode of action of the photoactivated, nonspecific toxin cercosporin in the generation of singlet oxygen has been known for over two decades (Daub and Ehrenshaft, 2000). Indeed, even the molecular basis for the protection of *Cercospora* and other fungi from the effects of cercosporin are being revealed. Thus, production of both an ABC transporter-like protein and elevated levels of pyrridoxal are important in the protection of *Cercospora* from the toxic effects of cercosporin (Daub and Ehrenshaft, 2000). Knowledge of these protectants is being used in strategies to generate transgenic plants with reduced phytotoxin sensitivity with a goal of reducing or preventing leaf spot disease. In addition to cercosporin, other classes of phytotoxins such as the beticolins from *C. beticola* have been shown to debilitate normal plant cell function (Goudet *et al.*, 1998). Beticolins are a family of polycyclic molecules sharing a common core structure but differing in the functional groups on the aromatic rings (Goudet *et al.*, 2000). Like cercosporin, the beticolins are known to destabilize membranes resulting in electrolyte leakage (Gapillout *et al.*, 1996). Additionally, the beticolins inhibit ATP-dependent proton transport (Simon-Plas *et al.*, 1996) and chelate magnesium (Mikès *et al.*, 1994). To date, the evidence suggests that phytotoxins produced by *Cercospora* species are virulence factors in plant infection. Enzymes reported to be secreted by

C. beticola as additional potential virulence factors include cellulase and pectinase (Pal and Mukhopadhyay, 1984; Srobarova and Brillova, 1979) and esterase (Weiland, 2001).

Host range, life cycle and aetiology

Symptomatic hosts of *C. beticola* include all members of *Beta* where they have been tested. In addition, many members of *Chenopodiaceae* exhibit leaf spot when inoculated with the pathogen. Reports of disease on many non-*Beta* plant species that have been attributed to *C. beticola*, however, often lack verification through pathogen isolation and inoculation to *B. vulgaris* (Chupp, 1953).

Natural inoculum of *C. beticola* in a sugarbeet field begins as stroma in infected leaf debris. It is postulated that sporulation may occur directly from overwintered stroma in organic matter or it may be preceded by saprophytic, vegetative growth of fungal mycelia. Once conidiation has commenced, water-splash, wind and insects are culprits in distributing spores on to leaf surfaces of the host (Lawrence and Meredith, 1970; Meredith, 1967; Pool and McKay, 1916), primarily on abaxial leaf surfaces. Because the abaxial surfaces of leaves possess a greater number of stomates than adaxial surfaces, the opportunity for invasion of leaf parenchyma through open stomates by an elongating hypha of *C. beticola* is maximized. Conidia deposited on host leaf, vein or petiole surfaces germinate under conditions of high humidity and leaf wetness and grow toward stomates during the pre-infection stage (Shane and Teng, 1983; Steinkamp *et al.*, 1979). An understanding of the optimum environmental conditions for the initiation of Cercospora epidemics (elevated temperature, humidity and leaf wetness; Bleiholder and Weltzien, 1972; Pool and McKay, 1916; Shane and Teng, 1983) has aided in the development of leaf spot prediction models for implementing fungicide spray schedules (see below).

Following penetration of the epidermis through the stomate (Rathaiah, 1977), fungal hyphae ramify the parenchymous tissue of these structures, growing intercellularly (Steinkamp *et al.*, 1979). Toxins then are produced in order to necrotize the cells in the vicinity of the branched hyphae. Nutrients are thus provided to the pathogen, and the necrotized tissue—primarily on the abaxial leaf surface—becomes the site of conidiophore and conidial development. Conidia again are dispersed by wind and rain splash to initiate new cycles of infection. In a controlled environment where sugarbeet is inoculated with *C. beticola* conidia, visible lesions appear on leaves at between 9 and 12 d.p.i. and 12 days is considered to approximate one sporulation cycle under field conditions. An interesting alternative route to the invasion of the sugarbeet plant by *C. beticola* has been proposed by Vereijssen *et al.* (2004): sugarbeet seedlings whose bare roots were exposed to fungal conidia succumbed to leaf spot disease several days after transplanting. This suggests that sugarbeet may support an endophytic or epiphytic phase of *C. beticola* vegetative growth prior to the induction of the pathogenic phase.

CHEMICAL CONTROL

As early as 1900, inorganic copper was being used by growers to control *C. beticola* on sugarbeet (Meriggi *et al.*, 2000). Not until over 50 years later did a systematic effort occur to develop fungicides with broad chemistries, efforts which have greatly benefited the beet sugar industry in the reduction of losses to leaf spot disease. Currently, both naturally derived and synthetic fungicides, both protectant and systemic, are available for *Cercospora* control (Ioannidis and Karaoglanidis, 2000). Although environmental health considerations limit the use of all available fungicide chemistries for disease control in sugarbeet, registration of compounds representing the major classes of fungicides by the environmental protection organizations of countries producing sugarbeet generally has occurred.

The availability of fungicides from different classes has become a crucial component in the control of Cercospora leaf spot on sugarbeet. Reports have been made of field isolates of *C. beticola* exhibiting resistance to fungicides in the benzimidazole class (Georgopoulos and Dovas, 1973; Ruppel and Scott, 1974; Weiland and Halloin, 2001) and increased tolerance to fungicides in the organotin (Bugbee, 1995; Cerato and Grassi, 1983) and triazole classes (Karaoglanidis *et al.*, 2000). This has prompted agriculturalists in sugarbeet production areas to promote fungicide rotation schedules in efforts to reduce the risk of selecting or enhancing fungicide-resistant strains of *C. beticola*. As a by-product of fungicide application research in sugarbeet, epidemiological models for the development of *C. beticola* epiphytotics have been constructed by several research teams for the prediction of disease severity and schedules for fungicide application (Shane and Teng, 1984; Windels *et al.*, 1998; Wolf and Verreet, 2002). These prediction models have resulted in a net decrease in fungicide use, simultaneously reducing the risk of fungicide resistance development and increasing the profitability of the crop to the producer.

RESISTANCE GENES AND MECHANISMS

Agronomical value

Sugarbeet varieties with resistance to leaf spot disease are now available in all countries of the world where *C. beticola* occurs regularly (Byford, 1996; Mechelke, 2000). The resistance conferred in these varieties is partial and strongest effects are seen under severe epidemics; under these conditions sugar yield as well as juice purity are significantly elevated in resistant varieties as compared with susceptible ones in the absence of fungicide use (Rossi, 1999). However, especially at late stages of a severe epidemic, host resistance is not sufficient to prevent damage on

the leaf canopy and disease-induced regrowth of new leaves resulting in a reduction in sugar yield (Rossi *et al.*, 2000). Fungicide applications do not always significantly further improve sugar yield and quality of the resistant varieties compared with fungicide spraying of the susceptible varieties only (Rossi *et al.*, 2000; Rossi, 1999). In countries like Greece and Italy with predictable severe leaf spot attack, yield losses can only be prevented by the use of resistant varieties plus fungicide application (Mechelke, 2000). When low to moderate disease severity occurs, however, high-yielding varieties treated with fungicide are favourable over resistant varieties, which continue to exhibit reduced yield compared with high-yielding varieties under disease-free conditions (Rossi, 1999). Nevertheless, under low to moderate occurrence of leaf spot, resistant varieties can delay spraying of fungicides and widen the interval time for fungicide application (Wolf and Verreet, 1997) when combined with field monitoring (Wolf and Verreet, 2002) and forecasting models (Racca *et al.*, 2002; Rossi and Battilani, 1991). Optimal disease management systems also have to consider simultaneous infection with powdery mildew and rust, which are controlled by most of the fungicides applied against *C. beticola* but not by genes conditioning leaf spot resistance (Mechelke, 2000; Ossenkop *et al.*, 2002; Rossi, 1999).

It has been noted that resistant varieties are lower yielding under disease-free conditions as compared with susceptible varieties (Mechelke, 2000; Miller *et al.*, 1994; Ossenkop *et al.*, 2002; Rossi, 1999). This may be due to (i) linkage drag of the donor chromosomes, (ii) the enormous breeding efforts needed to select for several resistance loci (Panella and Frese, 2000) without losing selection gain for yield performance (e.g. Mechelke, 2000) and (iii) the fact that some of the resistance genes may show epistatic interactions with those for sugar yield or could lead to reduced plant fitness in the absence of the pathogen. An extreme example has been described by Tao *et al.* (2000) for the *Rps2* gene of *Arabidopsis* for resistance to *Pseudomonas syringae*. Alternatively, the induced or permanent activation of the defence responses generating biochemical and morphological barriers (Feindt *et al.*, 1981a,b; Lieber, 1982) and/or the production of pathogenesis-related proteins is predicted to consume resources that might otherwise be used for the sugar production.

Source of host resistances

With respect to the development of plant agriculture, sugarbeet is a relatively new crop. Sugarbeet germplasm is poor in the diversity of known disease resistance genes. Historically, resistance was introgressed from the wild sea beet, *B. vulgaris* L. spp. *maritima* (Coons *et al.*, 1955; Hecker and Helmerick, 1985; Munerati, 1932). Resistant accessions can be found in compatible *B. vulgaris* subspecies (Asher *et al.*, 2001; http://www.genres.de/idb/beta/) and also in other sections of the genus *Beta*. Non-host resistance ranging from near incompatibility to extremely strong resistance are described among the *Beta* sections *Corollinae*, *Nanae* and *Procumbentes* (Carels *et al.*, 1990; Coons, 1975; Koch, 1985). Most of these species, unfortunately, are sexually incompatible with sugarbeet and not tractable for sugarbeet breeding (Gao and Jung, 2002; Mesbah *et al.*, 1997; Reamon-Ramos and Wricke, 1992).

Molecular basis of host resistance

Resistance to *Cercospora beticola* is polygenic and quantitative and the exact number of host genes contributing to the resistance remains unknown (Smith, 1987). A classical study of the complex inherited resistance by Smith and Gaskill (1970) suggests 4–5 major resistance genes involved in expression of leaf spot resistance in the crosses used. However, more genes are likely to contribute to resistance as examination of the entire germplasm collection continues. Histological studies of the host/pathogen interaction revealed various defence reactions of the plant after pathogen invasion (Feindt *et al.*, 1981a,b; Lieber, 1982). Some pathogenesis-related proteins with chitinase (Nielsen *et al.*, 1993) and glucanase (Gottschalk *et al.*, 1998) activity have been isolated that are speculated to be involved in host defence. One of a series of different antifungal proteins cloned from sugarbeet is described in Nielsen *et al.* (1997). Finally, low-molecular-weight metabolites such as tyramine (Harrison *et al.*, 1967) and phytoalexins (Martin, 1977) have been associated with defence against invasion by *C. beticola*.

Inheritance of host resistance

Reported broad-sense heritability estimates vary in a wide range from 0.12 to > 0.8, illustrating the quantitative nature of this trait. Different germplasm, population structures, environments, set-ups of the experiments and formula have been applied to this analysis. Some studies revealed higher heritability estimates ranging from > 0.8 (Schäfer-Pregl *et al.*, 1999; one F_2 and one related top-cross population, two environments, calculated with repeated measurements), and 0.80 (Koch and Jung, 2000; one F_2 population, microenvironments used for calculation) to 0.60–0.71 (Smith and Gaskill, 1970; three populations, 2 years, 40 replications each), whereas for others lower heritabilities from 0.24 (Smith and Ruppel, 1974; two populations), 0.17– > 0.8 (Saito, 1966; various populations, years, methods) to 0.12–0.16 (Bilgen *et al.*, 1969; two open-pollinated back-cross populations) were reported. These studies underline the difficulties in studying inheritance of resistance to *Cercospora beticola*, although all report that resistance is based mostly on additive components. Consequently, hybrid varieties can be composed of susceptible elite lines and donor lines for the resistance (Mechelke, 2000).

QTL mapping and molecular breeding

With the advent of molecular markers, a new way for the genetic localization of defence genes and the subsequent application in 'molecular breeding' was opened. Quantitative trait loci (QTL) mapping of polygenic inherited resistances is a powerful tool to unravel the number and positions of the genetic resistance factors on the linkage map of the host plant (Young, 1996). QTL to *Cercospora beticola* have been identified on all sugarbeet chromosomes (T. Kraft, personal communication): Nilsson *et al.* (1999) identified five QTL on four chromosomes, Schäfer-Pregl *et al.* (1999) seven QTL on six chromosomes, Koch and Jung (2000) four QTL on three chromosomes and Setiawan *et al.* (2000) also four QTL but located on four chromosomes. Most QTL were inherited through partially dominant to additive gene action, but always one or more recessive QTL were observed. In all studies, only a few major QTL with relatively short supporting intervals were observed, which is encouraging for the application of marker-assisted selection for Cercospora resistance in the future. A drawback comes from the observation of Schäfer-Pregl *et al.* (1999), who found one QTL only under artificial inoculation but not in the naturally infested field. Detailed studies of the host/pathogen interaction and extensive phenotyping in various environments will be needed before marker-assisted selection of resistance to *Cercospora beticola* will be reliably possible in the future.

Many simply inherited pathogen resistance genes have been cloned over the past decade (Jones, 2001). Most share common structural features (Dangl and Jones, 2001) making it possible to clone the analogous genes [resistance gene analogues (RGAs)] from other plant species, including sugarbeet (Koch, 2003). These RGAs can serve as candidates for mapped QTL, lending support to the reliability of the phenotyping and mapping and enabling a more directed cloning attempt of the QTL. This strategy was successfully applied to sugarbeet by Hunger *et al.* (2003), who identified RGA candidates within QTL intervals.

Host resistance components

Resistant varieties are effective against *C. beticola* in all sugarbeet growing areas of the world (Rossi, 1995; Ruppel, 1972; Smith, 1985; Smith and Campbell, 1996). Various resistant and susceptible varieties have been challenged with *C. beticola* in field observation trials and in controlled environment experiments in efforts to determine the components of the rate-reducing resistance to *Cercospora beticola*. Infection efficiency of conidia, incubation period, size of necrotic spots and spore yield, collectively, were significantly influenced for all of the resistant varieties tested that reduced disease severity. Not affected was the period until conidiophores produced from lesions and the rate of lesion expansion (Rossi *et al.*, 1999, 2000). With a simulation model, Rossi *et al.* (1999) could show that all resistance components will affect disease severity at about the same efficiency. Consequently, the fastest breeding progress can be achieved by selection on the resistance component with the largest variation available in the present germplasm. However, due to the easy spread of the conidia under a severe epidemic from plant to plant and plot to plot (Große-Herrenthey, 2001), neighbouring effects in plant variety trials will probably occur. Varieties with major effects based on reduction of the infection efficiency will be over-estimated at constant inoculum density and varieties with major effects on the spore yield will be underestimated. Ultimately, highest resistance levels are achieved by improving simultaneously all resistance components.

The example noted above of race C2 of *C. beticola* (Lewellen and Whitney, 1976; Solel and Wahl, 1971; Whitney and Lewellen, 1976), which is affected by the presence of the *Cb* resistance gene, constitutes the sole report of a hypersensitive-like resistance reaction to *C. beticola* in *B. vulgaris*. Because race C2 constitutes at best a minor component of *C. beticola* populations, however, the *Cb* gene was never deliberately implemented in breeding programmes (Skaracis and Biancardi, 2000), although it may constitute a QTL of the characterized quantitative resistance. Other virulences or host-specific pathotype interactions were often discussed but never reliably observed (for a summary see Rossi, 2000).

CONCLUSIONS AND FUTURE PROSPECTS

Cercospora leaf spot continues to be a serious impediment to sugarbeet production. Despite significant research and breeding efforts, fungicide application programmes, high-yielding sugarbeet varieties, or agronomic practices that guarantee leaf-spot-free sugarbeet production remain elusive to producers. In the meantime, the deployment of moderately resistant varieties combined with fungicide rotation, potentially integrating induced systemic resistance (Bargabus *et al.*, 2002, 2003), will be used in disease control for several years to come.

The potential for transgenic sugarbeet to be implemented for Cercospora leaf spot control has been considered by many research teams. Proprietary methods for gene transfer to sugarbeet have yielded breeding lines with engineered resistance to Rhizomania disease (Mannerlof *et al.*, 1996), caused by beet necrotic yellow vein virus, and resistance to herbicides (Bartsch and Pohl-Orf, 1996). Although the first generation of transgenic sugarbeet expressing antifungal proteins did not perform with the desired level of resistance to *C. beticola*, the future production of plants transgenic for genes encoding phytotoxin (e.g. cercosporin or beticolin) degrading enzymes (Mitchell *et al.*, 2002) or new optimized anti-fungal peptides (Gao *et al.*, 2000) may retard infection more effectively. Nevertheless, use of such varieties by producers at present will be delayed until the possible acceptance of genetically modified sugarbeet by consumers becomes a reality.

Asymptomatic hosts for plant pathogens can serve as pathogen reservoirs and for population build-up in fields rotated to a non-host crop (Agrios, 1988). Growth of *C. beticola* on non-host crops also would result in its exposure to fungicides of different classes, a possibility that has been discussed within the context of multiple fungicide resistance characterizing this organism. The use of polymerase chain reaction protocols for sensitive detection of pathogens has revolutionized the assay of asymptomatic hosts for the presence and quantity of target pathogens. Such methods currently are being tested for determining alternative hosts and asymptomatic hosts of *C. beticola* (Lartey *et al*., 2003).

Gene transfer methods based on direct DNA delivery to fungal protoplasts (Upchurch *et al*., 1994) or the incubation of conidiophore germlings with *Agrobacterium tumefaciens* (J. J. Weiland and J. T. Rasmussen, unpublished observations) promise to open new avenues for the investigation of pathogenesis and virulence in *C. beticola*. As the genomes of many phytopathogenic fungi are slated to be sequenced in the coming years, it is hoped that *C. beticola* will gain consideration as a 'priority pathogen' among such projects. Emerging biochemical (e.g. proteomic) and genome analysis (e.g. RNA silencing) techniques applied to *C. beticola* lay the foundation for an imminent fruitful era of study into this important disease of beet crops.

ACKNOWLEDGEMENTS

We thank A. Quinn of Betaseed, Inc. (Shakopee, MN) for permission to photograph in their leaf spot disease nursery, T. Friesen (USDA-Fargo) for the electrophoretic karyotype photograph, and T. Freeman (NDSU-Fargo) for electron micrographs of *C. beticola* lesions. All other photographs were produced by J.J.W.

REFERENCES

Agrios, G.N. (1988) *Plant Pathology*, 3rd edn. San Diego: Academic Press.

Anonymous (2003) *Sugar and Sweetener Situation and Outlook Yearbook*. Economic Research Service, U.S. Department of Agriculture, June 2003, SSS-2003.

Asher, M.J.C., Luterbacher, M.C. and Frese, L. (2001) Wild *Beta* species as a source of resistance to sugar-beet pests and diseases. *Int. Sugar J.* **103**, 447–456.

Bargabus, R.L., Zidack, N.K., Sherwood, J.E. and Jacobsen, B.J. (2002) Characterisation of systemic resistance in sugar beet elicited by a non-pathogenic, phyllosphere-colonizing *Bacillus mycoides*, biological control agent. *Physiol. Molec. Plant Path.* **61**, 289–298.

Bargabus, R.L., Zidack, N.K., Sherwood, J.E. and Jacobsen, B.J. (2003) Oxidative burst elicited by *Bacillus mycoides* isolate Bac J, a biological control agent, occurs independently of hypersensitive cell death in sugar beet. *Molec. Plant–Microbe Interact.* **16**, 1145–1153.

Bartsch, D. and Pohl-Orf, M. (1996) Ecological aspects of transgenic sugar beet: transfer and expression of herbicide resistance in hybrids with wild beets. *Euphytica*, **91**, 55–58.

Bilgen, T., Gaskill, J.O., Hecker, R.J. and Wood, D.R. (1969) Transferring Cercospora leaf spot resistance from *Beta maritima* to sugarbeet by backcrossing. *J. Am. Soc. Sugar Beet Techn.* **15**, 444–449.

Bleiholder, H. and Weltzien, H.C. (1972) Beiträge zur Epidemiologie von *Cercospora beticola* Sacc. & Zuckerrübe. II: Die Konidienbildung in Abhängigkeit von den Umweltbedingungen Temperatur, relative Luftfeuchtigkeit und Licht. *Phytopath. Z.* **73**, 46–68.

Bugbee, W.M. (1995) *Cercospora beticola* tolerant to triphenyltin hydroxide. *J. Sugar Beet Res.* **30**, 167–174.

Byford, W.J. (1996) A survey of foliar diseases of sugar beet and their control in Europe. In *Proceedings of the 59th IIRB Congress*. Brussels: International Institute for Beet Research, pp. 1–10.

Carels, N., Dekegel, D., van Heule, G. and Lepoivre, P. (1990) Symptomatological and morphological study of the resistance of wild beet species of the *Patellares* section to *Cercospora beticola* Sacc. *J. Phytopathol.* **130**, 17–330.

Cerato, C. and Grassi, G. (1983) Tolerance of organo-tin compounds among *Cercospora beticola* isolates. *Informatore Fitopatologico*, **33**, 67–69.

Chupp, C. (1953) *A Monograph of the Fungus Genus Cercospora*. Ithaca, NY.

Coons, G.H. (1975) Interspecific hybrids between *Beta vulgaris* L. and the wild species of *Beta*. *J. Am. Soc. Sugar Beet Techn.* **18**, 281–306.

Coons, G.H., Owen, F.V. and Steward, D. (1955) Improvement of the sugar beet in the United States. *Adv. Agron.* **7**, 89–139.

Dangl, J.L. and Jones, J.D.G. (2001) Plant pathogens and integrated defence responses to infection. *Nature*, **411**, 826–833.

Daub, M.E. and Ehrenshaft, M. (2000) The photoactivated *cercospora* toxin *cercosporin*: contributions to plant disease and fundamental biology. *Annu. Rev. Phytopathol.* **38**, 461–490.

Duffus, J.E. and Ruppel, E.G. (1993) Diseases. In *The Sugarbeet Crop* (Cooke, D.A. and Scott, R.K., eds). London: Chapman & Hall, pp. 346–427.

Feindt, F., Mendgen, K. and Heitefuss, R. (1981a) Feinstruktur unterschiedlicher Zellwandreaktionen im Blattparenchym anfälliger und resistenter Rüben (*Beta vulgaris* L.) nach Infektion durch *Cercospora beticola* Sacc. *Phytopath. Z.* **101**, 248–264.

Feindt, F., Mendgen, K. and Heitefuss, R. (1981b) Der Einfluss der Spaltöffnungsweite und des Blattalters auf den Infektionserfolg von *Cercospora beticola* bei Zuckerrüben (*Beta vulgaris* L.) unterschiedlicher Anfälligkeit. *Phytopath. Z.* **101**, 281–297.

Gao, A.G., Hakimi, S.M., Mittanck, C.A., Wu, Y., Woerner, B.M., Stark, D.M., Shah, D.M., Liang, J. and Rommens, C.M. (2000) Fungal pathogen protection in potato by expression of a plant defensin peptide. *Nat. Biotechnol.* **18**, 1307–1310.

Gao, D. and Jung, C. (2002) Monosomic addition lines of *Beta corolliflora* in sugar beet: plant morphology and leaf spot resistance. *Plant Breed*, **121**, 81–86.

Gapillout, I., Mikès, V., Milat, M.L., SimonPlas, F., Pugin, A. and Blein, J.P. (1996) *Cercospora beticola* toxins. Part XIV: use of fluorescent cyanine dye to study their effects on tobacco cell suspensions. *Phytochemistry*, **43**, 387–392.

Georgopoulos, S.G. and Dovas, C. (1973) Occurrence of *Cercospora beticola* strains resistant to benzimidazole fungicides in northern Greece. *Plant Dis. Report*, **62**, 321–324.

Goodwin, S.B., Dunkle, L.D. and Zismann, V.L. (2001) Phylogenetic analysis of *Cercospora* and *Mycosphaerella* based on the internal transcribed spacer region of ribosomal DNA. *Phytopathology*, **91**, 648–658.

Gottschalk, T.E., Mikkelsen, J.D., Nielsen, J.E., Nielsen, K.K. and Brunstedt, J. (1998) Immunolocalization and characterization of a beta-1,3-glucanase from sugar beet, deduction of its primary structure and nucleotide sequence by cDNA and genomic cloning. *Plant Sci.* **132**, 153–167.

Goudet, C., Milat, M.L., Sentenac, H. and Thibaud, J.B. (2000) Beticolins, nonpeptidic, polycyclic molecules produced by the phytopathogenic fungus *Cercospora beticola*, as a new family of ion channel-forming toxins. *Mol. Plant–Microbe Interact.* **13**, 203–209.

Goudet, C., Véry, A.-A., Milat, M.-L., Ildefonse, M., Thibaud, J.-B., Sentenac, H. and Blein, J.-P. (1998) Magnesium ions promote assembly of channel-like structures from beticolin 0, a non-peptide fungal toxin purified from *Cercospora beticola*. *Plant J.* **14**, 359–384.

Große-Herrenthey, U. (2001) *Molekulare Analyse der genetischen Variabilität von* Cercospora beticola *Sacc., dem Erreger der Cercospora-Blattfleckenkrankheiten bei Zuckerrüben, und Untersuchungen zur Resistenz von* Beta vulgaris *ssp. maritima gegen* C. beticola *unter Anwendung unterschiedlicher Resistenztestsysteme*. PhD thesis, Christian-Albrechts-Universität Kiel, Germany.

Harrison, M., Maag, G.W., Payne, M.G., Hecker, R.J. and Remmenga, E.E. (1967) Sampling for 3-hydroxytyramine and polyphenol oxidase in sugar beets. *J. Am. Soc. Sugar Beet Techn.* **14**, 470–479.

Hecker, R.J. and Helmerick, R.H. (1985) Sugar-beet breeding in the Unites States. In *Progress in Plant Breeding—1* (Russell, G.E., ed.). London: Butterworths, pp. 37–61.

Hunger, S., Di Gaspero, G., Möhring, S., Bellin, D., Schäfer-Pregl, R., Borchardt, D.C., Durel, C.E., Werber, M., Weisshaar, B., Salamini, F. and Schneider, K. (2003) Isolation and linkage analysis of expressed disease-resistance gene analogues of sugar beet (*Beta vulgaris* L.). *Genome*, **46**, 70–82.

Ioannidis, P.M. and Karaoglanidis, G.S. (2000) Resistance of *Cercospora beticola* Sacc. to fungicides. *Adv. Sugar Beet Res.* IIRB, **2**, 123–147.

Jones, J.D.G. (2001) Putting knowledge of plant disease resistance genes to work. *Curr. Opin. Plant Biol.* **4**, 281–287.

Karaoglanidis, G.S., Ioannidis, P.M. and Thanassoulopoulos, C.C. (2000) Reduced sensitivity of *Cercospora beticola* to sterol-demethylation-inhibiting fungicides. *Plant Pathol*, **49**, 567–572.

Koch, F. (1985) Integrierte Bekämpfung der Blattfleckenkrankheit (*Cercospora beticola*) unter Berücksichtigung der Resistenzzüchtung. In *Proceedings of the 48th IIRB Congress*. Brussels: International Institute for Beet Research, pp. 307–331.

Koch, G. (2003) GABI-BEET: the German sugar beet genome initiative. In *Proceedings of the 1st Joint IIRB-ASSBT Congress, 27 February–1 March 2003, San Antonio, TX USA*, pp. 249–260.

Koch, G. and Jung, C. (2000) Genetic localization of *Cercospora* resistance genes. *Adv. Sugar Beet Res.* **IIRB**, 2, 197–210.

Lartey, R.T., Weiland, J.J., Caeser-TonThat, T.-C. and Bucklin-Comiskey, S. (2003) A PCR protocol for rapid detection of *Cercospora beticola* in sugarbeet tissues. *J. Sugar Beet Res.* **40**, 1–10.

Lawrence, J.S. and Meredith, D.S. (1970) Wind dispersal of conidia of *Cercospora beticola*. *Phytopathology*, **60**, 1076–1078.

Lewellen, R.T. and Whitney, E.D. (1976) Inheritance of resistance to race C2 of *Cercospora beticola* in sugarbeet. *Crop Sci.* **16**, 558–561.

Lieber, H. (1982) *Untersuchungen zur Erfassung der ResistenZ. der Zuckerrübe,* Beta vulgaris *L., gegenüber.* Cercospora Beticola. *Sacc.* PhD thesis, Georg-August—Universität Göttingen, Germany.

Mannerlof, M., Lennerfors, B.-L. and Tenning, P. (1996) Reduced titer of BNYVV in transgenic sugar beets expressing the BNYVV coat protein. *Euphytica*, **90**, 293–299.

Martin, S.S. (1977) Accumulation of flavadnoids betagarin and betavulgarin in *Beta vulgaris* infected by the fungus *Cercospora beticola*. *Physiol. Plant Path.* **11**, 297–303.

Mechelke, W. (2000) Züchtungs- und Sortenstrategien zur ResistenZ. bei Zuckerrüben gegenüber *Cercospora beticola*. *Zuckerindustrie*, **9**, 688–692.

Meredith, J.S. (1967) Conidium release and dispersal in *Cercospora beticola*. *Phytopathology*, **57**, 889–893.

Meriggi, P., Rosso, F., Ioannides, P.M. and Ayala Garcia, J. (2000) Fungicide treatments against Cercospora leaf spot in sugar beet (*Beta vulgaris* L.). *Adv. Sugar Beet Res.* **IIRB**, 2, 77–102.

Mesbah, M., Scholten, O.E., deBock, T.M. and Lange, W. (1997) Chromosome localisation of genes for resistance to *Heterodera schachtii*, *Cercospora beticola* and *Polymyxa betae* using sets of *Beta procumbens* and *B. patellaris* derived monosomic additions in *B. vulgaris*. *Euphytica*, **97**, 117–127.

Mikès, V., Milat, M.L., Pugin, A. and Blein, J.P. (1994) *Cercospora beticola* toxins. Part VII: fluorometric study of their interactions with biological membranes. *Biochim. Biophys. Acta*, **1195**, 124–130.

Miller, J., Rekoske, M. and Quinn, A. (1994) Genetic resistance, fungicide protection and variety approval policies for controlling yield losses from Cercospora leaf spot infection. *J. Sugar Beet Res.* **31**, 7–12.

Mitchell, T.K., Chilton, W.S. and Daub, M.E. (2002) Biodegradation of the polyketide toxin cercosporin. *Appl. Environ. Microbiol.* **68**, 4173–4181.

Munerati, O. (1932) Sull'incrocio della barbabietola coltivata con la beta selvaggia della costa adriatica. *L'industria Saccarifera Italiana*, **25**, 303–304.

Nielsen, K.K., Mikkelson, J.D., Kraugh, K.M. and Bojsen, K. (1993) An acidic class III chitinase in sugar beet: induction by *Cercospora beticola*, characterization, and expression in transgenic tobacco plants. *Mol. Plant–Microbe Interact.* **6**, 495–506.

Nielsen, K.K., Nielsen, J.E., Madrid, S.M. and Mikkelsen, J.D. (1997) Characterization of a new antifungal chitin-binding peptide from sugar beet leaves. *Plant Physiol.* **113**, 83–91.

Nilsson, N.O., Hansen, M., Panagopoulos, A.H., Tuvesson, S., Ehlde, M., Christiansson, M. and Rading, I.M. (1999) QTL analysis of Cercospora leaf spot resistance in sugar beet. *Plant Breed*, **118**, 327–334.

Ossenkop, A., Ladewig, E. and Manthey, R. (2002) Performance of Cercospora resistant varieties—consequences for variety testing and advisory service. *Zuckerindustrie*, **127**, 867–871.

Pal, V. and Mukhopadhyay, A.N. (1984) Study of the cellulolytic and pectolytic enzymes in nine biological forms of *Cercospora beticola* Sacc. *Acta Phytopathol. Acad. Sci. Hung.* **19**, 263–269.

Panella, L. and Frese, L. (2000) Cercospora resistance in *Beta* species and the development of resistant sugar beet lines. *Adv. Sugar Beet Res.* IIRB, 2, 163–176.

Pool, V.W. and McKay, M.B. (1916) Relation of stomatal movement to infection by *Cercospora beticola*. *J. Agric. Res.* **5**, 1011–1038.

Racca, P., Jörg, E., Mittler, S. and Petersen, J. (2002) Sugarbeet leaf diseases—Forecasting approaches for the optimization of fungicide application. *Zuckerindustrie*, **127**, 949–958.

Rathaiah, Y. (1977) Stomatal tropism of *Cercospora beticola* in sugarbeet. *Phytopathology*, **67**, 358–362.

Reamon-Ramos, S.M. and Wricke, G. (1992) A full set of monosomic addition lines in *Beta vulgaris* from *Beta webbiana*: morphology and isozyme markers. *Theor. Appl. Genet.* **84**, 411–418.

Rossi, V. (1995) Effect of host resistance in decreasing infection rate of *Cercospora* leaf spot epidemics on sugarbeet. *Phytopathologia Med.* **34**, 149–156.

Rossi, V. (1999) Effect of host resistance and fungicide sprays against Cercospora leaf spot in different sugar beet-growing areas of the Mediterranean basin. *Phytopathologia Med.* **38**, 76–88.

Rossi, V. (2000) Cercospora leaf spot infection and resistance in sugar beet (*Beta vulgaris* L.). *Adv. Sugar Beet Res.* **IIRB**, 2, 17–48.

Rossi, V. and Battilani, P. (1991) Cercopri: a forecasting model for primary infections of *Cercospora* leaf spot of sugarbeet. *OEPP/EPPO Bull.* **00**, 527–531.

Rossi, V., Battilani, P., Chiusa, G., Giosue, S., Languasco, L. and Racca, P. (1999) Components of rate-reducing resistance to Cercospora leaf spot in sugar beet: incubation length, infection efficiency, lesion size. *J. Plant Pathol.* **81**, 25–35.

Rossi, V., Battilani, P., Chiusa, G., Giosue, S., Languasco, L. and Racca, P. (2000) Components of rate-reducing resistance to Cercospora leaf spot in sugar beet: conidiation length, spore yield. *J. Plant Pathol*, **82**, 125–131.

Rossi, V., Giosue, S. and Racca, P. (1999) A model integrating components of rate-reducing resistance to Cercospora leaf spot in sugar beet. *J. Phytopathol*, **147**, 339–346.

Ruppel, E.G. (1972) Variation among isolates of *Cercospora beticola* from sugarbeet. *Phytopathology*, **62**, 134–136.

Ruppel, E.G. and Scott, P.R. (1974) Strains of *Cercospora beticola* resistant to benomyl in the USA *Plant Dis. Report*, **58**, 434–436.

Saccardo, P.A. (1876) Fungi Veneti novi vel critici. Series V. *Nuovo Giornale Bot. Italiano*, **8**, 162–211.

Saito, K.-I. (1966) Studies on the Cercospora leaf spot resistance in sugar beet breeding. *Mem. Fac. Agric.* **6**, 113–179.

Schäfer-Pregl, R., Borchard, D.C., Barzen, E., Glass, C., Mechelke, W., Seitzer, J.F. and Salamini, F. (1999) Localization of QTLs for tolerance to *Cercospora beticola* on sugar beet linkage groups. *Theor. Appl. Genet.* **99**, 829–836.

Setiawan, A., Koch, G., Barnes, S.R. and Jung, C. (2000) Mapping quantitative trait loci (QTLs) for resistance to Cercospora leaf spot disease (*Cercospora beticola* Sacc.) in sugar beet. *Theor. Appl. Genet.* **100**, 1176–1182.

Shane, W.W. and Teng, P.S. (1983) Epidemiology of Cercospora leafspot 1982. *Sugarbeet Res. Ext. Rept.* **13**, 201–213.

Shane, W.W. and Teng, P.S. (1984) *Cercospora beticola* infection prediction model 1983. *Sugarbeet Res. Ext. Rept.* **14**, 174–179.

Simon-Plas, F., Gomes, E., Milat, M.L., Pugin, A. and Blein, J.P. (1996) *Cercospora beticola* Toxins (X. Inhibition of Plasma Membrane H+-ATPase by Beticolin-1). *Plant Physiol.* **111**, 773–779.

Skaracis, G.N. and Biancardi, E. (2000) Breeding for Cercospora resistance in sugar beet. *Adv. Sugar Beet Res.* **IIRB**, 2, 177–196.

Smith, G.A. (1985) Response of sugarbeet in Europe and the USA to *Cercospora beticola* infection. *Agron. J.* **77**, 126–129.

Smith, G.A. (1987) Sugar beet. In: *Principles of Cultivar Development: Crop Species* (Fehr, W.R., ed.). New York: Macmillan, pp. 577–625.

Smith, G.A. and Campbell, L.G. (1996) Association between resistance to Cercospora and yield in commercial sugarbeet hybrids. *Plant Breed*, **115**, 28–32.

Smith, G.A. and Gaskill, J.O. (1970) Inheritance of resistance to Cercospora leaf spot in sugarbeet. *J. Am. Soc. Sugarbeet Techn.* **16**, 172–180.

Smith, G.A. and Ruppel, E.G. (1974) Heritability of resistance to *Cercospora beticola* leaf spot in sugarbeet. *Crop Sci.* **14**, 113–115.

Solel, Z. and Wahl, I. (1971) Pathogenic specialization of *Cercospora beticola*. *Phytopathology*, **61**, 1081–1083.

Srobarova, A. and Brillova, D. (1979) Study of the effect of Cercospora disease of beet on the polysaccharides of the host cell wall. *Pol'nohopodarstvo*, **25**, 878–886.

Steinkamp, M.P., Martin, S.S., Hoefert, L.L. and Ruppel, E.G. (1979) Ultrastructure of lesions produced by *Cercospora beticola* in leaves of *Beta vulgaris*. *Physiol. Plant Path.* **15**, 13–26.

Tao, Y., Yuan, F.H., Leister, R.T., Ausubel, F.M. and Katagiri, F. (2000) Mutational analysis of the *Arabidopsis* nucleotide binding site-leucine-rich repeat resistance gene RPS2. *Plant Cell*, **12**, 2541–2554.

Upchurch, R.G., Meade, M.J., Hightower, R.C., Thomas, R.S. and Callahan, T.M. (1994) Transformation of the fungal soybean pathogen *Cercospora kikuchii* with the selectable marker. *Bar. Appl. Envir. Micro.* **60**, 4592–4595.

Vereijssen, J., Schneider, J.H.M. and Termorshuizen, A.A.J. (2004) Possible root infection of *Cercospora beticola* in sugarbeet. *Eur. J. Plant Path.* **110**, 103–106.

Weiland, J.J. (2001) Light-induced secretion of esterase by *Cercospora beticola* Sacc. In *10th International Congress of Molecular Plant Microbe Interaction*. St Paul MN: IS-MPMI (abstract).

Weiland, J., Eide, J., Rivera-Varas, V. and Secor, G. (2001) Genetic diversity of *Cercospora beticola* in the U.S. and association of molecular markers with tolerance to the fungicide triphenyltin hydroxide (TPTH). *Phytopathology*, **91**, 94 (abstract).

Weiland, J.J. and Halloin, J.M. (2001) Benzimidazole resistance in *Cercospora beticola* sampled from sugarbeet fields in Michigan, USA. *Can. J. Plant Path.* **23**, 78–82.

Whitney, E.D. and Lewellen, R.T. (1976) Identification and distribution of races C1 and C2 of *Cercospora beticola* from sugarbeet. *Phytopathology*, **66**, 1158–1160.

Windels, C.E., Lamey, H.A., Hilde, D., Widner, J. and Knudsen, T. (1998) A Cercospora leaf spot model for sugar beet: in practice by an industry. *Plant Dis.* **82**, 716–726.

Wolf, P.F.J. and Verreet, J.A. (1997) Epidemiological development of *Cercospora beticola* (Sacc.) in sugar beet crops. *J. Plant Dis. Protect.* **104**, 545–556.

Wolf, P.F.J. and Verreet, J.A. (2002) An integrated pest management system in Germany for the control of fungal leaf diseases in sugar beet: the IPM sugar beet model. *Plant Dis.* **86**, 336–343.

Young, N.D. (1996) QTL mapping and quantitative disease resistance in plants. *Annu. Rev. Phytopathol.* **34**, 479–501.